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# Targeting focal adhesion assembly by ethoxyfagaronine prevents lymphoblastic cell adhesion to fibronectin

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| Résumé en anglais | **Background:** Leukemic cell adhesion to proteins of the bone marrow microenvironment provides signals which control morphology, motility and cell survival. We described herein the ability of ethoxyfagaronine (etxfag), a soluble synthetic derivative of fagaronine, to prevent leukemic cell adhesion to fibronectin peptide (FN/V).<br>**Methods:** Phosphorylation of fak and pyk2 were evaluated by immunoblotting. Labelled proteins were localized by confocal microscopy. PI 3-kinase activity was evaluated by in vitro kinase assay.<br>**Results:** Subtoxic concentration of etxfag reduced L1210 cell adhesion to FN/V dependently of β1 integrin engagement. Etxfag impaired FN-dependent formation of β1 clustering without modifying β1 expression at the cell membrane. This was accompanied by a decrease of focal adhesion number, a diminution of fak and pyk2 phosphorylation at Tyr-576, Tyr-861 and Tyr-579, respectively leading to their dissociations from β1 integrin and inhibition of PI 3-kinase activity. Etxfag also induced a cell retraction accompanied by a redistribution of phosphorylated fak and pyk2 in the perinuclear region and lipid raft relocalization.<br>**Conclusion:** Through its anti-adhesive potential, etxfag, combined with conventional cytotoxic drugs could be potentially designed as a new anti-leukemic drug. |
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